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☐ 1: M69043. Homo sapiens MAD-...[gi:187290]

Links

LOCUS HUMMAD3A 1550 bp mRNA linear PRI 07-MAR-1994
DEFINITION Homo sapiens MAD-3 mRNA encoding Ikb-like activity, complete cds.
ACCESSION M69043
VERSION M69043.1 GI:187290
KEYWORDS
SOURCE Homo sapiens (human)
ORGANISM Homo sapiens
Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE 1 (bases 1 to 1550)
AUTHORS Haskill, S., Beg, A.A., Tompkins, S.M., Morris, J.S., Yurochko, A.D.,
Sampson-Johannes, A., Mondal, K., Ralph, P. and Baldwin, A.S. Jr.
TITLE Characterization of an immediate-early gene induced in adherent
monocytes that encodes I kappa B-like activity
JOURNAL Cell 65 (7), 1281-1289 (1991)
MEDLINE 91292530
PUBMED 1829648
COMMENT Original source text: Homo sapiens cDNA to mRNA.
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Exhibit A

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```

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187290	1	Jul 26 1993 19:32	Dead
187290	1	Apr 27 1993 12:57	Dead

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Accession M69043 was first seen at NCBI on Apr 27 1993 12:57

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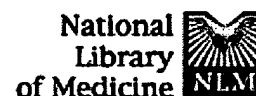
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☐ 1: Proc Natl Acad Sci U S A. 1993 Mar 15;90(6):2532-6.

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Mutual regulation of the transcriptional activator NF-kappa B and its inhibitor, I kappa B-alpha.

Brown K, Park S, Kanno T, Franzoso G, Siebenlist U.

Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.

The NF-kappa B transcription factor complex is sequestered in the cytoplasm by the inhibitory protein I kappa B-alpha (MAD-3). Various cellular stimuli relieve this inhibition by mechanisms largely unknown, leading to NF-kappa B nuclear localization and transactivation of its target genes. It is demonstrated here with human T lymphocytes and monocytes that different stimuli, including tumor necrosis factor alpha and phorbol 12-myristate 13-acetate, cause rapid degradation of I kappa B-alpha, with concomitant activation of NF-kappa B, followed by a dramatic increase in I kappa B-alpha mRNA and protein synthesis. Transfection studies reveal that the I kappa B-alpha mRNA and the encoded protein are potently induced by NF-kappa B and by homodimers of p65 and of c-Rel. We propose a model in which NF-kappa B and I kappa B-alpha mutually regulate each other in a cycle: saturating amounts of the inhibitory I kappa B-alpha protein are destroyed upon stimulation, allowing rapid activation of NF-kappa B. Subsequently, I kappa B-alpha mRNA and protein levels are quickly induced by the activated NF-kappa B. This resurgence of I kappa B-alpha protein acts to restore an equilibrium in which NF-kappa B is again inhibited.

PMID: 8460169 [PubMed - indexed for MEDLINE]

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